ABSTRACT

The present invention provides a GLAST knockout mouse lacking the function of an endogenous glutamate transporter GLAST gene, which shows: 1) an intraocular pressure within the normal range; and 2) a reduction in the number of cells in the retinal ganglions when compared with a wild-type normal mouse. Owing to the ocular properties, this knockout mouse is useful as a model for normal tension glaucoma. By using this knockout mouse, a compound useful for the treatment of normal tension glaucoma can be screened.